LONG-TERM RESULTS OF SURGERY FOR ACQUIRED CARDIOVASCULAR DISEASE

*Transcription of a Panel Meeting**

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MODERATOR CHARLES E. KOSSMANN: The modern era of heart surgery began approximately ten years ago, and in view of this, the Committee on Medical Education of the Academy felt that the time had come for an appraisal of the results of this type of surgery in terms of a decade of follow-up.

We shall begin by asking a few questions which will, perhaps, interest you as well as the various members of the Panel. First, we shall talk about valvular disease, since this is the big area of modern-day heart surgery. If we have time, we shall discuss the long-term results of surgery for other types of acquired cardiovascular disease. The valve which has received the most attention is, of course, the mitral valve.

I shall ask Dr. Ellis to present briefly the long-term overall results of commissurotomy in the first 1,000 cases which, I believe, were all, or mostly all, operated upon at the Boston City Hospital by his associate, Dr. Dwight Harken.

DR. LAURENCE B. ELLIS: I think I can show this best by slide (Fig. 1). I doubt very much if this series of patients differs in any appreciable degree from the results obtained by other surgeons, but it happens to be a consecutive series operated by one surgeon or his immediate associates, and we now have a follow-up extending over about ten years. This represents the first 1,000 patients. On the left are the Group II and III patients, on the right Group IV. These correspond roughly to the American Heart Association's classification, and for our purposes today, I think they can be considered essentially the same. You can see that these represent the status at each year of follow-up of the survivors of the operation. At the end of one year, some 84 per cent of Group III patients were improved, and the improvement tends to decrease as the years go by. The size of the group at seven to eight years is small, so it may not be statistically valid, and the actual drop in improvement may be less than appears. In Group IV about 40 per cent are improved at the end of eight years.

The important points are: that there is a substantial number of patients who are improved and who maintain their improvement, and conversely; that the persistence of improvement tends to diminish as the years go by, which is, of course, only to be expected, since this is a palliative operation and not a curative one; and that a surprisingly large number still remain improved at the end of a certain period of time.

MODERATOR KOSSMANN: We can use these data as a springboard from

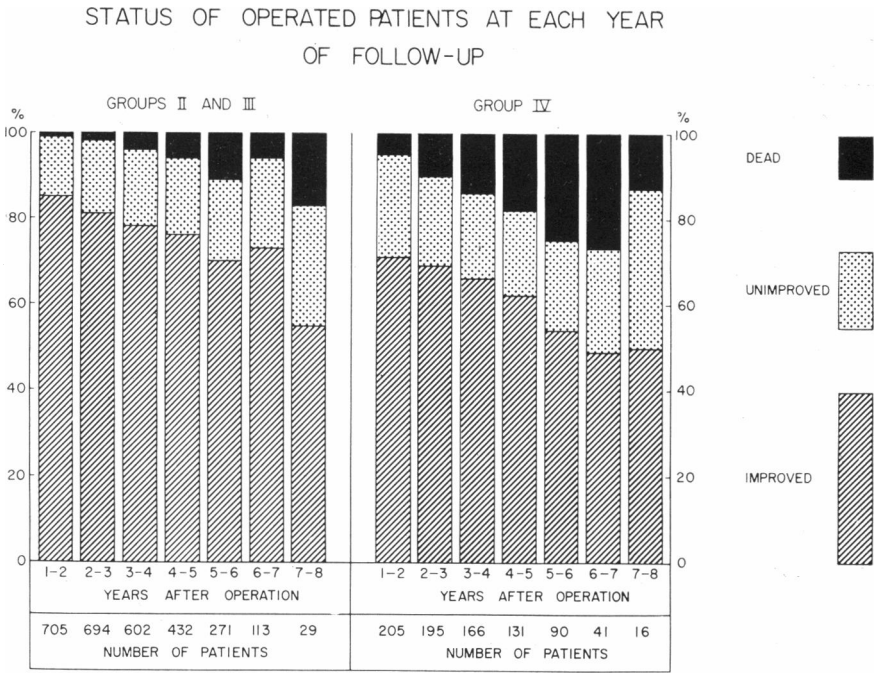

Figure 1.—From: Harken, D. E., Black, H. and Ellis, L. B., Clinical study of 1,000 consecutive cases of mitral stenosis two to nine years after mitral valvuloplasty, *Circulation 19*:803-20 (809). Reproduced by permission of the authors and the American Heart Association, Inc.

which to jump into other aspects of the long-term results of commissurotomy. As in any surgical procedure, these results will naturally depend, in great part, on the technical excellence and thoroughness of the surgeon. I would like to ask Dr. Johnson to discuss the technical variables which influence the results of mitral commissurotomy.

DR. JULIAN JOHNSON: Obviously, the result that is to be obtained by the use of this procedure depends greatly upon the status of the patient selected for operation, and upon how thoroughly the operation is carried out. You have seen the results which Dr. Harken has obtained in his first 1,000 cases. There is no question that we do a much better job now than we used to do when commissurotomy was first started. I think most surgeons were hesitant about tackling the posterior commissure if it were calcified. If we were successful in opening the anterior commissure, but found the posterior commissure calcified, most of us were content to leave it alone. The patients were improved temporarily.

As time went on, the heart still had to work against a partial obstruction, and eventually the patient deteriorated. We shall go more deeply into that problem later on.

If the valve is of such a nature that it can be opened completely without producing regurgitation, there is no question that the patient is benefited tremendously. One of the difficulties with the operation of commissurotomy is that one cannot see the site of operation, as it is commonly practiced by the closed technique. No one knows what is going on, except the surgeon who has his finger in the heart, and in many cases, *he* does not know what is going on, so that it takes a great deal of experience to know exactly what has occurred and how best to carry out the opening of the valve. Many of the original papers on this subject stated that mitral commissurotomy was not worth while. I am sure the reason for this was that the surgeons who were doing the operation did not really succeed in getting the valve open. For example, I know that one of America's great surgeons, when doing a commissurotomy several years ago, said, "Now this valve is so small that I cannot get my finger through it", and he pushed his finger through further and said, "Now I have gotten it open so that I can get it over my first knuckle. I am not going to kill this woman. I shall leave her alone, just leave the valve as it is." You and I know that that is no way at all to get the mitral valve open. We think the mitral valve ought to be opened so that there is, preferably, a three-finger opening. There is no question that, as the cardiac surgeon gains experience, he learns more precisely how widely the valve can be opened, and he is more apt to succeed. But I also think that, since the know-how has become rather universally spread throughout the country, the major problem now—at least with the surgeon who has done several hundred or more mitrals—is how to get some of these badly calcified valves open so that they function again. In some of them it seems almost impossible to accomplish this.

Even though we are still doing most of them by the closed technique, it would seem obvious that a valve could be opened more accurately under direct vision. Whether we shall get to the point of doing all of our mitrals on the heart-lung machine I am not sure. At the present time, we are doing some of ours that way, especially those in which the valves are very heavily calcified, and some that we are redoing because we were not able to open them satisfactorily the first time. You may be amused to know that with one recent patient, when

I looked down at the mitral valve directly, I could not recognize anything about it, it was so badly calcified. I had to close my eyes and feel it to ascertain where the commissures were located! I had not looked at very many valves but had felt a great many. It would seem obvious, however, that with experience one could do better with the open technique.

MODERATOR KOSSMANN: *Dr. Johnson, does the opposite technical aspect sometimes have an effect, namely, opening the valve too widely?*

DR. JOHNSON: I don't believe there is such a thing as opening the valve too widely. The problem arises from opening the valve in an area not in the commissure. If that occurs a mitral regurgitation may develop. I can speak only for myself, and that is to say that I am, perhaps, a little on the cautious side and have had very few people who have developed mitral regurgitation of any real significance. I am probably much more likely not to have opened the valve as widely as I should have, rather than having produced regurgitation.

MODERATOR KOSSMANN: Dr. Ellis, from the study of your series, I think you have some ideas on this from the medical point of view. *Did you find mitral insufficiency a common complication of commissurotomy?*

DR. ELLIS: In the first place, speaking of what Dr. Johnson was saying, in studying a group of patients who deteriorated, we found a surprisingly large number of patients who had done so; that is, people who first improved and then became worse, had mitral insufficiency. In some, the insufficiency was present at the time of the first operation; in others it was only discovered at the time of the second operation that mitral insufficiency had been produced or had been increased by the first procedure. As a group, these people have done much less well than the ones who had pure stenosis. So, in the long run, mitral insufficiency seems to be a poor thing to have. With the laudable zeal of the surgeons to get the valve wide open, there is a certain definite danger that some of them will be opened in the wrong place, with the resulting insufficiency, and this must be guarded against.

MODERATOR KOSSMANN: Dr. Johnson?

DR. JOHNSON: I would agree that the patient who has some regurgitation originally may have the regurgitation increased when the valve is opened. An occasional one will have decreased insufficiency as the valve is mobilized, but the patient who has no regurgitation preoperatively

is not very likely to develop significant regurgitation postoperatively; at least in our hands it has been a small number. It will depend a good deal, I suppose, on the technique used. I know Dr. Harken uses his finger whenever possible. I would suspect that he produces very little regurgitation in the patient who did not have it beforehand.

MODERATOR KOSSMANN: Can you state a figure on that, Dr. Ellis? *How many of the patients do you think show eventual deterioration as a result of mitral insufficiency produced or aggravated by the surgery?*

DR. ELLIS: We have only three guides as to what has happened. First, what the surgeon sees at the time of the first operation, that there is more insufficiency after than before the valvotomy. This is only a rough guide because the patient's blood pressure sometimes is very low at this point in the operation, and it is hard to gauge properly the regurgitation quantitatively. The second guide is to reoperate the patient and find there is a considerable degree of insufficiency which was previously not suspected or believed to be significant, or to have a post-mortem examination, and the latter is a very poor guide as to the presence of insufficiency. The third guide is, of course, the clinical evaluation on the basis of murmurs and other clinical changes, or possibly by some of the special tests. These, however, are not too accurate in quantitatively distinguishing insufficiency from stenosis. I cannot give offhand the exact number of patients who had insufficiency, either at the time of surgery, or aggravated by surgery; but of 220 who deteriorated after improvement, insufficiency occurred in 20 or so per cent. Beyond that I cannot say.

MODERATOR KOSSMANN: I would like to leave the technical aspects at this time. Obviously, the long-term results will be determined by the proper selection of the patient, and I think our audience would like to know whether one can actually select for commissurotomy the patient who will have the best long-term results, on the basis of the ordinary history, physical examination and laboratory data. Would you like to try that, Dr. Harvey?

DR. RÉJANE M. HARVEY: I think that we would agree that one certainly can, on the basis of clinical findings alone—history, physical examination, x-rays and electrocardiogram—select the patient who will benefit from surgery. However, I feel that if one used the criteria which we would demand if we were to use only these means we would be excluding a certain number of patients who would also benefit from

surgery, but about whom we might have some doubts or reservations. For example, if a patient has a history of dyspnea of relatively short duration, without objective evidence of peripheral congestion coupled with the isolated murmur of mitral stenosis, minimal to moderate enlargement of the pulmonary artery, of the left atrium and right ventricle, in whom the electrocardiogram shows a sinus mechanism, and not too much in the way of a right hypertrophy pattern, that patient will have a very good immediate result from surgery. But I also feel that by using such rigid criteria I would exclude patients who on repeat examinations, or on other examinations, might also be expected to have fairly good results.

MODERATOR KOSSMANN: *Are there any comments you would care to make on selection, Dr. Ellis?*

DR. ELLIS: I agree that the patients with pure mitral stenosis, those who have not been in congestive failure, are the ones who are likely to do the best, and I should think that, with some experience, one could select 90 per cent of those on clinical grounds, with about as high a degree of accuracy as one can obtain with added catheterization results. The other 10 per cent, especially patients in chronic failure or those in whom associated insufficiency is present, are the patients that are much harder to assess. In them catheterization and similar tests give some help, but often not the critical help that we would like, because the tests may be deficient in the exact areas where we want information. It is important, however, to be able to recognize mitral stenosis in the presence of failure, for we still obtain substantial improvement in the Group IV patients. These, in general, are the patients who have had recurrent heart failure, and even at the end of eight or nine years, 40 or more per cent of those surviving operation are improved.

DR. JOHNSON: *Do any of you have any figures as to how long those Group IV patients would have lived had they not been operated upon?*

DR. ELLIS: It is hard to find comparable figures of medically followed patients. In spite of the tremendous number of studies on mitral disease, there are very few series that have been studied in exactly the same way, taking symptomatic patients right from the time the symptoms are developed. The Oleson figures from Denmark are probably the best, and, in general, agree with other studies. In this respect, all Group IV patients were dead at the end of seven years. We find that 55 or 60 per cent of the Group IV patients who have surgery are alive after nine and

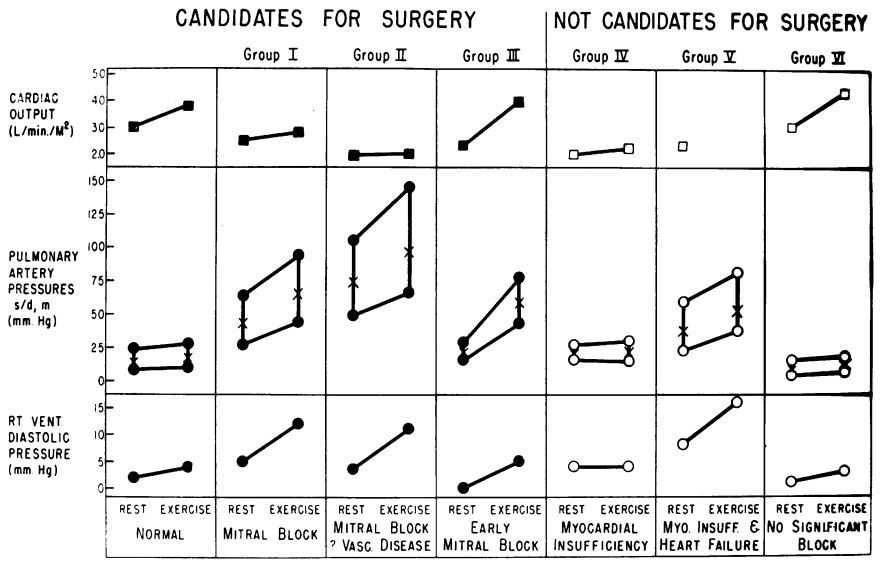

Figure 2.—From: Harvey, Réjane M. and Ferrer, M. Irené, Consideration of hemodynamic criteria for operability in mitral stenosis and in mitral insufficiency, *Circulation 20*:442-50, 1959. Reproduced by permission of the authors and the American Heart Association, Inc.

ten years. The interesting thing is that the survival rate of the Group IV patients, after surgery, even discounting the surgical mortality, actually isn't very different from the Group III patients. This survival of 55 per cent takes into account the 20-odd per cent who died at operation.

DR. JOHNSON: As I looked at your chart it was 40 per cent.

DR. ELLIS: This was improvement. This chart did not include survival.

DR. JOHNSON: This is only improvement?

DR. ELLIS: About 40 per cent of that group who survived operation are improved after nine or ten years, but if you add the operative mortality it would decrease the percentage.

DR. JOHNSON: Mr. Chairman, I think there should not be any shilly-shallying about this. The truth of the matter is: if you can get the mitral valve open without regurgitation, the patient is going to be improved, no matter what his original condition.

MODERATOR KOSSMANN: We may have a little difference of opinion on that.

DR. JOHNSON: The only problem is to get the patient through the operation, and to open the valve without giving him regurgitation.

MODERATOR KOSSMANN: We would like to discuss this very point a little bit more in detail. Coming back to the matter of selection, *I would like to ask Dr. Harvey whether the patients who are most likely to benefit from surgery have a characteristic hemodynamic profile?*

DR. HARVEY: (Slide—Fig. 2) This is a hemodynamic sketch, if you will, of some of the patterns that have been encountered in patients with the isolated finding of mitral stenosis. There is no one in these groups with a systolic murmur, enlarged left ventricle, nor any patient who was found to have a regurgitant jet at the time of surgery. The first pattern is represented by a patient who has mitral stenosis and a considerable degree of pulmonary hypertension at rest, aggravated by exercise, with a relatively fixed cardiac output, that is, it does not increase normally with exercise. This is the group that was described originally by Dexter and his colleagues in Boston, which they have shown by hemodynamic studies, as well as by Dr. Ellis's clinical studies, to have had very good improvement following surgery. This is the group of which I gave the clinical picture earlier. I do think these people can be picked out on clinical grounds alone. We have our best results in this group.

In the second group, we have those who have severe pulmonary hypertension, which sometimes can reach rather frightening levels. The mortality in this group is high in most reported series. The ones that have been operated on have had a good result from a hemodynamic point of view, but one rarely finds a drop in pulmonary artery pressures to as low a level as may occur in the first group.

The third group has bothered us considerably. These are a group of patients that in our experience at Lenox Hill Hospital and at Bellevue Hospital are found in increasing numbers. This third pattern is found in the patient who has a history of rheumatic heart disease and mitral stenosis, who has periods of disability with unusual stress, pregnancy, or severe physical exertion. They have normal pulmonary artery pressures at rest which rise very sharply on exercise, reaching levels not unlike those found at rest in the first group. The cardiac output moves up rather briskly. It seemed to us that this group of patients should be the ones who would really have the best result from surgery, because at this stage they have so little pulmonary hypertension at rest that the

pulmonary vascular bed is probably not much involved. We have had some good results in this group, but we have also had some disappointing results which stem from technical difficulties. The left atrial appendages in these patients are often so small that the surgeons have difficulty in entering with a knife and must resort to exploration with their fingers. The valves are long and leathery and not always amenable to finger fracture. We wondered whether this group might not do better under open-heart surgery.

The next group includes those patients who have normal pressures at rest, which cannot rise on exercise, and who have a fixed cardiac output. We, and others, have operated on these patients. We have not changed their clinical course, or the hemodynamic picture. I personally do not believe that these patients are suitable subjects for commissurotomy as the primary problem rests in the myocardium and not in the mitral valve.

The next group of patients is one that Dr. Ellis mentioned previously, the patients with intractable heart failure. To find a patient with mitral stenosis who has no other lesions, a normal left ventricle, yet intractable heart failure, is not common, but we *have* seen it. They have a moderate to severe pulmonary hypertension, a low fixed cardiac output, and an elevated right ventricular diastolic pressure. We have also operated on them in the past, but do not do so any longer. Although they have survived immediate operation, there has been absolutely no clinical or hemodynamic evidence of improvement up to the time of death, some time about two years postoperative.

The last group is represented by those patients—and it would be interesting to know whether Dr. Johnson would consider operating on them—who have the physical findings of mitral stenosis, a normal electrocardiogram and only minor changes in the size of the pulmonary artery and left atrium. At Bellevue we have not operated on this group. Dr. Kossmann's group at Lenox Hill has, and perhaps he would like to discuss them.

MODERATOR KOSSMANN: You can show my first slide, as Dr. Harvey has asked me to discuss it (Slide—Fig. 3). This is one of the patients that Dr. Harvey mentioned. We were rather interested in this group, because at rest and on exercise they show normal dynamics. The chart is similar to the one Dr. Harvey had, but the important things are the cardiac output and the pulmonary artery pressure. These are normal

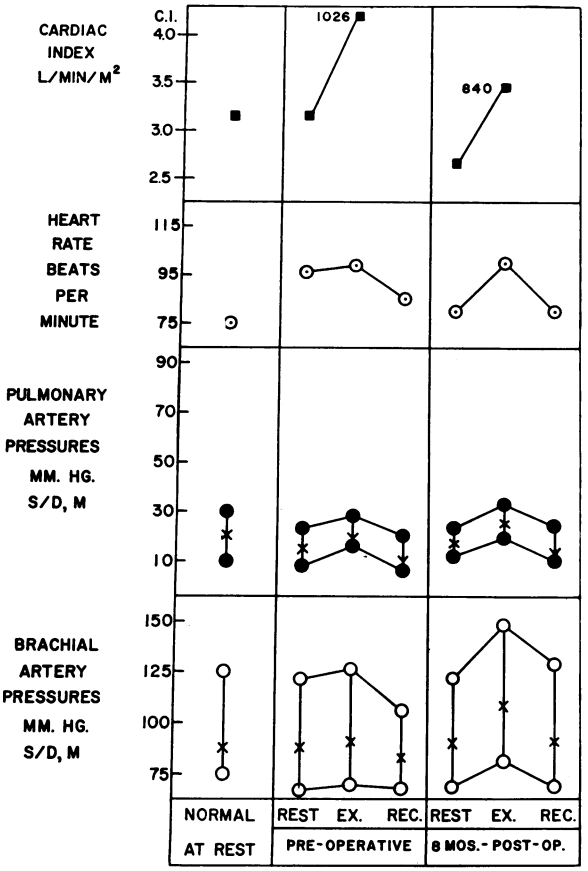

Figure 3.—Pt. E.C., female, age 43, rheumatic mitral stenosis with normal hemodynamics at rest and on exercise.

The cardiac index, heart rate, and direct pressures in the pulmonary and brachial arteries are shown. In the first vertical column are the normal values; in the second the values obtained in the patient at rest, immediately after exercise, and after recovery from the exercise; and in the third column the same data obtained 8 months after attempted commissurotomy. The figures on the cardiac outputs with exercise (1026 and 840) indicate the increase in cardiac output in ml per 100 ml increase in 0_2 consumption per minute. The oxygen consumption in each instance was approximately doubled by exercise.

At operation the mitral valva orifice easily admitted a finger and a half. Its rubbery consistency and elongated conical form made finger fracture technically impossible. The mean gradient of pressure measured across the mitral valve at operation was 8 mm. Hg. The hemodynamics were still normal but flows at rest and exercise were smaller than before operation. (Data collected and chart prepared by Dr. Francis X. Claps.)

values in this patient. The pulmonary diastolic and systolic pressures are normal and on effort do not budge, but the cardiac output goes up considerably. In the recovery period there is little change. This particular patient had an episode, two years earlier, of hemoptysis and

enlarged liver. The story certainly sounded very much like heart failure, yet when we saw her, although the symptoms indicated that there was heart impairment, the dynamics, as judged by the cardiac output and the pulmonary artery pressures, were nevertheless normal.

In order to learn whether anything could be achieved in such a patient, we proceeded to operate on her, because we know that in many clinics the clinical manifestations in such cases are accepted as sufficient justification for surgery. When the surgeon got in he found that the valve admitted only a finger and a half. Also, the valve was rubbery in consistency and he could not accomplish anything with his finger alone. Further, we measured the gradient of pressure across the valve at operation. It was small, the mean being in the neighborhood of 8 mm. of mercury. This, of course, was unchanged by the operation. The patient continued to have symptoms. She was studied again eight months later, and the only change in the dynamics at that time was that the cardiac output in response to effort, although still within the normal range, had fallen somewhat. In each case the amount of exercise given was such as to increase the oxygen consumption to twice the resting value. So there was in each study a comparable amount of exercise.

This group is interesting; first, in that it is questionable whether one could do very much for the dynamic situation which is normal to begin with. Second, if you insist that any gradient across the valve in diastole is important and must be corrected, then I would think that in this type of case it cannot be done by simple finger fracture. Third, it is also interesting that this woman has shown some deterioration post-operatively, if you can accept the decrease in response to exercise as evidence of deterioration, without any obvious reason, such as rheumatic activity, upper respiratory infection, embolization, pregnancy, etc.

The patient is one of a group in which we ought to get a little more information on what we are achieving when we operate on them.

Dr. Johnson, one of your remarks raised a question a little earlier. *Do you think these two presentations answer that question about opening of the mitral valve being certain to give an increase in flow?*

DR. JOHNSON: I do not think anything you have said has shown that it does not. In the last case that you demonstrated, you did not open the mitral valve, so there is no argument there. I would certainly agree that there is a lot to the problem of rheumatic heart disease with mitral stenosis, aside from the size of the valve opening. Perhaps I am a little

premature in bringing up this topic, but I am sure every surgeon has seen patients who were very poor surgical risks, yet had valves that were not nearly as tight as some of those found in others who were considered good risks. I think that the duration and extent of the disease may have a good deal to do with such a condition. For example, last week I operated on a relatively young woman in her late thirties, who had a relatively small heart and whom I considered an excellent operative risk. Nevertheless, at operation her mitral valve was as tight as any I have ever felt. I am sure this kind of patient can be operated upon with a mortality of not more than 1 or 2 per cent. Had that patient not been operated upon at this time but carried along on a medical regimen, as might well have been the case, in a semi-invalid state for the next ten years, I would hypothesize that her heart would then be very large and she would be a very poor-risk patient, although her mitral valve opening would be (I imagine) the same size, because it could hardly have become any smaller. So, there is a great deal to the problem besides the exact size of the valve opening. On the other hand, everything else being equal, I cannot see that it is not a good thing to have the valve opened. If mitral stenosis is not present in the first place it does no good to operate on the patient.

MODERATOR KOSSMANN: Dr. Ellis, you have been strangely silent in this little controversy here. How about some remarks?

DR. ELLIS: I would like to make a comment in connection with one thing Dr. Harvey said about patients with intractable failure. They were cardiac invalids who required the most rigorous treatment to keep them out of severe failure.

DR. JOHNSON: Could I have a definition of intractable failure?

DR. ELLIS: A person who cannot be kept dry reasonably well—and I don't mean getting his liver down to normal size, but who can be well dried out and who can at least be made fairly comfortable.

DR. JOHNSON: Would you not include a large liver as intractable failure?

DR. ELLIS: No, I would not necessarily include such patients in Group IV.

DR. JOHNSON: Here there are two experts on the subject and they cannot agree what intractable failure is. So you see the problem involved is agreeing on terms. We operate upon a good many patients whom we consider intractable heart failures, including those with a

large liver. To these desperately sick patients, whom we consider intractable failures, we frequently may quote a risk as high as 50 per cent at the time of operation.

MODERATOR KOSSMANN: The Chairman rules that a persistently large, congested liver, despite adequate therapy, means intractable failure! Will you go on from there, Dr. Ellis?

DR. ELLIS: I would agree in general, Dr. Johnson, except that there is also a matter of definition of what is improvement. Of course the people whom we show as improved in these charts are patients who are substantially improved. There is a small group with mitral stenosis who have a valve that is not helped very much. Some of them probably have quite severe pulmonary vascular changes that are irreversible, although we have been happily surprised, at least in the clinical results, to find how well such patients do.

Dr. Johnson cites the patient who may be in heart failure ten years from now. This may or may not be due to mitral stenosis. At that point it is true that we do not obtain good results.

MODERATOR KOSSMANN: I would conclude that there certainly is a difference of opinion on the significance of the hemodynamic profile in the patient with mitral stenosis. Actually, there can be no disagreement on the existence of such differences, because there are great variations in the behavior of the circulation in mitral stenosis. This undoubtedly depends not only on the degree of stenosis, but on other factors such as involvement of the myocardium, the pulmonary vasculature, and others we don't know about. I would gather from the discussion that this is an area which could still be studied profitably because usually differences of opinion arise from a paucity of quantitative data.

I would now like to go back to a technical problem. Dr. Johnson, I was interested in your remarks about not ever seeing the valve and having to feel your way in the bottom of that little bowl of blood in the left atrium. I wanted to ask whether you feel that restenosis is a real thing, is a real problem? I ask this because in looking at these valves at necropsy they are sclerotic or fibrotic, avascular and very often calcified. It is difficult to see how any kind of inflammatory reaction could occur in the late stages of the disease. *Would you make some comments on restenosis, and give us your feeling on this matter about which we talk so glibly?*

DR. JOHNSON: There is a good deal of difference of opinion around

the country as to whether these valves do restenose. I do not think there is any doubt that if the valve stenoses in the first place, it can stenose again, particularly if the patient has another bout of rheumatic fever. How often that occurs is another question. We in Philadelphia do not like to operate on young people, and the primary reason is that we think they are more likely to have recurrent attacks of rheumatic fever and destroy the results of the operation. If we can avoid the operation in the young we do so. I personally do not think that restenosis is any great problem. I think that the person whom we select as the ideal candidate is one who does not have calcification of the valves and whose heart is not tremendous to start with, even though the pulmonary artery pressure may be high. If the valve can be opened completely, or nearly completely, I have not the slightest fear that that valve will restenose, unless the patient has another episode of rheumatic fever. On what basis do we make this judgment? It is merely on our own experience. We have reoperated upon 12 or 15 of those on whom we did the first operation. Most of these we operated upon from the left side originally, and then from the right side, as a secondary procedure. Of these 15 there are only two in whom I am convinced that there was restenosis. In the others I am sure I just did not get the valve open completely at the first operation. Back in '47-'48, when we started, we were not opening the posterior commissure very often, particularly if it was calcified. We opened the anterior commissure. The patient would hold up for five years and then regress to the state in which he was preoperatively. We do the second operation from the right side and take the risk of opening the calcified posterior commissure. Some of these patients have been markedly improved after the second operation. In some, we have dislodged a calcific embolus and they have died at the time of the operation. At least one patient came through the operation, but we got a good bit of regurgitation and he died as a result of that in six months or so. From our own experience, we believe that actual restenosis is a rare occurrence rather than a common one, and that most of the difficulty has arisen from failure to effect a complete opening of the valve at the first operation.

MODERATOR KOSSMANN: *Dr. Ellis, do you have some views on this?*

DR. ELLIS: I am in agreement with Dr. Johnson. Actually, it is a very difficult problem, because about the only good criteria for restenosis are the availability of descriptions of both first and second operations

done by the same surgeon. The vast majority of patients we see require a second operation, and some of those examined post-mortem after the first operation, have had an inadequate job done the first time either because, as Dr. Johnson said, only one commissure was opened or the valve was so sclerotic, so calcified, that it was virtually immobile. In a great many of these early operations no attempt was made to open the fused chordae, which is standard procedure now. I think I have seen half a dozen patients who have had completely adequate openings made at the first operation and had a tight stenosis within a matter of four or five years. Only one or two of these had clinical rheumatic fever in the interim.

MODERATOR KOSSMANN: *Dr. Harvey, have you any experience with restenosis?*

DR. HARVEY: We think we have now seen two. In neither instance has this yet been confirmed by surgery. It has been confirmed by a measurement of pulmonary artery pressures. I can cite one case. This patient was a young woman, 21 years of age, who had an extremely stormy pregnancy. One month following delivery she had a cardiac catheterization performed. She had a sinus mechanism, did not have a very large heart, and had an isolated murmur of mitral stenosis. The pressures of the pulmonary artery were very high. The cardiac output was at the lower limits of normal. She had a commissurotomy performed, and clinically and hemodynamically we were very well pleased. She had a very striking drop in pulmonary artery pressures, with no change in blood flow. The electrocardiograms had shown a very marked right hypertrophy pattern in leads from the right precordium, with inversion of T waves. One year postoperative, these T waves were upright and the R wave in the right precordial leads diminished in amplitude. We continued to follow her, and about three years postoperative it was noted that the T waves were becoming inverted and the R waves in the right precordial leads were increasing in size. It was difficult to get this patient to admit to any change in her clinical status. It was obvious to us that she was beginning to deteriorate. Her pulmonary artery pressures three years following her operation were as high as they had been preoperatively. I would have to say she had restenosed, although in the absence of clinical evidence of rheumatic fever, the reason for restenosis is obscure.

DR. JOHNSON: Has she been reoperated upon?

DR. HARVEY: This study was done six weeks ago. We had one other in whom the change took place not at a month, not at one year, not at three years but at five years after operation.

DR. JOHNSON: Why are you waiting? Why don't you get that girl operated on?

DR. KOSSMANN: *Dr. Harvey, is there any possibility that this pressure in the pulmonary artery is caused by changes in the pulmonary vasculature itself rather than in the mitral valve?*

DR. HARVEY: I think, when one sees pressures as high as these, that one has to invoke some change in pulmonary vasculature. Most of us are aware of the fact that even in their presence, as Dr. Ellis said earlier, one can still have very striking changes in pressure. Just what mechanism is responsible for the return of severe pulmonary hypertension, we do not know. There is a possibility that the patient is having intravascular thrombosis.

MODERATOR KOSSMANN: Let us move along. I would like to bring up the problem of embolization as a factor in determining the long-term results, and *I would like to have just a word said about late peripheral embolization. I think you have had some experience, Dr. Ellis.*

DR. ELLIS: Again referring to this group of a thousand patients, of whom 900-odd survived operation, we have an excellent follow-up, and of this entire group to date, with an average follow-up period of about four years, at least half of these patients are fibrillating. Twenty per cent of them had been on long-term anticoagulants. As far as we know, only 30 patients have had major late peripheral emboli. Although it is hard to prove statistically, still there is a striking difference. We think operation does confer substantial protection against embolization in patients with mitral stenosis of some degree of severity.

MODERATOR KOSSMANN: I have no questions from the audience, so I shall go on to the matter of mitral insufficiency. Surgery, of course, has been performed for this anatomical defect for some time, and it looks as though we might be in a position to try to make some evaluation of the long-term results. *First, I would like to ask Dr. Harvey whether the physiologic understanding of mitral insufficiency has any bearing on the results of surgical intervention?*

DR. HARVEY: I shall try to be very brief about this. I think that one thing we should realize is that the hemodynamic consequences of experimentally, acutely induced mitral insufficiency are not the same as

the hemodynamic findings in the symptomatic patient with mitral insufficiency. Three different explanations have been offered to explain the discrepancy between the experimental and the clinical findings.

Burchell of the Mayo Clinic has suggested that the reason that patients who have mitral insufficiency develop pulmonary congestion is because the left ventricle is overloaded during diastole and that, as a consequence of this, the ventricle fails. Hence they develop the picture one associates with the usual type of left ventricular failure. Dexter in Boston and Wood also, of the Mayo Clinic, have suggested that the reason for finding evidences of pulmonary congestion in patients with mitral insufficiency is that mitral stenosis is also always present. When there is mitral stenosis, of course, pulmonary congestion can result. The third suggestion, which stems from experimental work, is that the left ventricle must always be embarrassed by some means other than initial insufficiency, before the valvular lesion becomes important. Whether this is due to an associated aortic valvular lesion, or whether it is the result of some injury to the myocardium itself, we don't know. I think the experience of some surgeons, who have been using the open-heart technique, tends to confirm the belief that the actual valvular lesion *per se* may not be the underlying problem. I am thinking particularly of the published reports by Varca, by Scott, and their associates, who have occasionally found that the mitral valve was intact and that it was the annulus which was enlarged. As you know, most of us do not think of annular dilation *per se* as being a consequence of rheumatic mitral insufficiency.

MODERATOR KOSSMANN: Here we might again consider the matter of technique. *Dr. Johnson, has any technique that you know of given superior long-term results? Are the surgeons agreed on any one particular technique at the moment?*

DR. JOHNSON: I don't think we have a really good operation as yet for mitral regurgitation. I have just seen the type of patient that Dr. Harvey was referring to, in whom the valve was certainly incompetent and the atrium tremendous and the annulus was large. The valves themselves seemed normal and perhaps thin. What caused the disease I am not sure. Why do these people go into failure? They fail because the pump does not work. The valve is leaking, just as my fruit sprayer does not work when it backfires. You can say it as scientifically as you wish, but if the valve does not keep blood in the left ventricle so it can go

into the aorta, it does not work very well. I really don't think we have a very good operation for this. We are operating on these patients in the open heart, attempting to close them down enough so that their function can be improved, and we have helped some, while others have not been helped. One of the great difficulties is that, when done by the open procedure, it is difficult to know when the regurgitation has been corrected. If the heart is closed and you have your finger in the atrium you can feel the jet coming up, and you know when you have accomplished something, whether you have succeeded in eliminating the regurgitation. The silly part of it is that, in many cases, you can put your finger behind the heart, push up on the annulus posteriorly, and stop the regurgitation completely. It seems ridiculous that one cannot do something to overcome this defect easily. A Nickel's operation done in Bailey's clinic would occasionally do a very good job. The circumferential suture used by Glover and his group has done well. We have had a few cases of that kind who have done extraordinarily well. We have a few where we did well, with the open-heart machine, by suturing the annulus under direct vision. By and large, I think our problems may be that, from the first, we have not felt that the operation is a good one. Therefore we don't do it readily, except on people who already are poor operative risks. So our mortality has been high. We don't really feel sufficiently enthusiastic about it, however, to operate on cases who, despite regurgitation, are not in too bad shape. The time may come when we shall have more confidence in it and can operate on these patients early. I am not sure. Many times, as one looks at these badly calcified, very defective valves, one cannot see how in the world they can be made to work again. What we are doing now is to operate on them under direct vision. The leak is usually at the posterior commissure, due to the loss of valve substance. As a rule, we put a suture in the annulus posteriorly, to pull the leaflets closer together. The best result we have had was in a patient who had mild aortic regurgitation, so that enough blood leaked back into the left ventricle, and we could tell when we had corrected the mitral valve completely.

MODERATOR KOSSMANN: *Dr. Ellis, do you think the surgery of mitral insufficiency will ever approach in quality the results obtained in properly selected cases of mitral stenosis?*

DR. ELLIS: I think the answer to that stems directly from what Dr. Harvey said. It is always a problem of a combination of mitral stenosis

and insufficiency in varying degrees. Pure mitral insufficiency is a "rare bird". In the occasional case where there is destruction of leaflets from ulcerative endocarditis, one might correct that, just as one can correct a primum defect, with excellent results. Often, insufficiency is produced mainly by dilation of the annulus. In this condition, the myocardial element is very important. By the time the patients become symptomatic, no matter how much one corrects the insufficiency, you will not get at the root of the matter. Actually, in our group of patients operated on by the closed technique for mitral stenosis alone, the patients who turned out to have an associated degree of marked insufficiency at the time of surgery, have, as a group, done just as well as patients operated on by various operations specifically designed to correct mitral insufficiency. However, the duration of improvement is not as a rule very long-lasting.

MODERATOR KOSSMANN: *I would now like to go on to aortic stenosis, and as long as you are talking, Dr. Ellis, would you continue? Are there any good criteria to be used in the selection of patients for aortic valvotomy for aortic stenosis?*

DR. ELLIS: With aortic stenosis, from the point of view of the clinical set-up, we are dealing with a condition somewhat different from mitral stenosis. It is a condition in which the medical prognosis is very ominous, once these patients become symptomatic. I am referring to isolated calcific aortic stenosis. For that reason, the physician does not have very long to decide whether to select surgical or medical treatment. Also, these patients are much poorer risks at the present time, by any of the operative techniques used, and the whole clinical picture can be completely mimicked by predominant coronary disease, with minimal aortic stenosis, including calcified aortic valves. For that reason I feel much more strongly about patients with aortic stenosis than I do with most mitrals. Before undergoing surgery they should have thorough catheterization studies because one frequently cannot evaluate the aortic stenosis without it.

MODERATOR KOSSMANN: *I wonder if you have encountered the problem, as reported from the National Heart Institute, of finding a gradient across the valve by catheter, but not finding any aortic stenosis at operation?*

DR. ELLIS: I regret to say that we do have a patient currently in the hospital, who had a 50 mm. gradient across the valve, in whom the

surgeon, Dr. Harkens, could not find any substantial degree of aortic or subaortic narrowing.

MODERATOR KOSSMANN: *Dr. Johnson, would you again tell us the preferred technique in this kind of disease, and what the immediate mortality of valvotomy is for isolated aortic stenosis?*

DR. JOHNSON: I think here again the technique varies in a good many parts of the country, and it is difficult to know which of these will turn out to be the one employed universally, say ten years from now. It would seem reasonable that anyone could do an operation better if he were looking at the valve directly, than if he were doing the operation blindly. So it is hard to believe that the open procedure will not win out in the end. However, at the present time, we are doing them blind again. We did them blind at first, and then we did them by finger fracture. Then we did them on the open-heart machine, and now we have gone back to doing them blind again, simply because we felt we were not doing much better with these calcified valves when looking at them, than when dilating them with the aid of a dilator. I hasten to add that we have not practiced decalcification of the aortic valves, trying to perform the same procedure on the aortic valve that one would in an artery by endarterectomy. There are a few clinics where the surgeons are trying to remove the calcium from the superior surface of the valve. If that can be done, leaving a normally functioning valve behind, of course it certainly sounds good. However, as was to be expected, in the preliminary efforts the mortality has been high. At the present time, the procedure which we are using is the small dilator of Mr. Brock inserted through the left ventricle. Formerly, when we used the dilator through the left ventricle, the difficulties encountered were largely related to the development of arrhythmia at the time of operation, and to the ability of the heart to absorb the shock of the procedure as we went through it. So now we put our patients on the heart machine, but *not* on the lung machine. In other words, we do a left-sided cardiac bypass, taking the blood out of the left atrium and putting it into the femoral artery, with the left ventricle at rest. So we don't have to put in any purse string sutures, and there is very little bleeding. Since the blood is being sucked out of the atrium, we can open the valve slowly, deliberately, and not subject the heart to the shock it might have received from more rapid opening. We think we are doing better by this technique, although time alone will give us the answer to that. We

have been able to do this with a mortality in the neighborhood of five per cent.

MODERATOR KOSSMANN: *In acquired or congenital?*

DR. JOHNSON: We do not do it in the congenital types, only for the acquired condition. We do all the congenitals by the open technique. There you are dealing with a different problem. In just trying to tear them open with a dilator you may get uncontrolled regurgitation, and in the congenital lesions you have, as a rule, a much better valve to work on and you can open the commissures under direct vision. If you don't become too enthusiastic and try to open too widely and get regurgitation, you do very well with the congenital group. We have not done a large number; 15 congenitals without a mortality, with some regurgitation in two or three of the early ones, when we were a little too enthusiastic and tried to open the commissure completely. Since learning that we should not go too close to the aortic wall, we have done pretty well with these cases. At the moment, we have given up doing the acquired calcific disease under direct vision, simply because we thought our pressure readings were just as good after using the small dilator. There is no doubt in my mind that, within a few years, we shall be putting in new aortic valves because the present state of affairs is highly unsatisfactory. In some of these patients one cannot see the commissures. One just has to guess where they should be and then make a bicuspid valve out of a hole in a calcified valve. That is the reason I decided they looked so bad I would rather not look at them to begin with until we can really think about replacing the valve completely. It is a very suitable area in which to place valves as compared with the mitrals. We may get to putting new valves in mitrals as well. I cannot see any reason for not putting synthetic valves in the aortic area.

MODERATOR KOSSMANN: I want to check on one figure. *Did you say "immediate" mortality of acquired aortic stenosis was five per cent?*

DR. JOHNSON: Yes, that is correct with this new technique that we are using. As a matter of fact, Dr. Glover in Philadelphia had the same mortality when he did not use the left bypass. We were never able to accomplish it without the left-heart bypass. When we used the Brock dilator without the left-heart bypass, we had a mortality in the neighborhood of 15 to 20 per cent. Since we have been doing it by the left-heart bypass—and I will admit our series is not large—we have had only

one death in 19 operations. Of course the mortality may go up with further experience, I admit that. But in any event the whole procedure, as far as the stress of the operative procedure is concerned, is tremendously relieved by the left-heart bypass. The heart just does not look as though it has "gone through the wringer", as it does when it has to work at the same time one is opening the aortic valve.

MODERATOR KOSSMANN: *Do you perfuse the coronaries?*

DR. JOHNSON: We don't open the aorta the way we are now doing it. It is closed. We take the blood out of the left atrium and put it into the femoral by means of a pump. The patient is heparinized. Then we open the valve blindly with the Brock dilator through the left ventricle. The left ventricle is relatively flaccid, has a low pressure, and bleeds very little. So there is no problem for the heart. It behaves beautifully, as if one were not doing anything to it, whereas if it has to work while one is operating on it, that is where we get into trouble. Although Dr. Glover succeeded, with a 5 per cent mortality even under those circumstances, we were never able to equal it.

MODERATOR KOSSMANN: *You do not interfere with the coronary circulation?*

DR. JOHNSON: We do not interfere with it at all.

MODERATOR KOSSMANN: *Do you have any long-term data?*

DR. JOHNSON: As a matter of fact, Dr. Harken's figures were essentially the same by the superior approach, coming down through the aorta. We have not been able to get as good figures with the approach from above as we have from below.

DR. ELLIS: (Slide) There are not many good figures available. Obviously, from what Dr. Johnson has said, there have been a number of different techniques used and it is very difficult to compare them, and the follow-up is not very long or large. Fortunately, this condition is not as common as mitral disease. This slide shows the follow-up of 86 consecutive patients done by the closed technique; the total operative mortality was about 20 per cent, but in the last 60 cases the mortality has dropped to 10 per cent. These represented—

DR. JOHNSON: I am sure Dr. Harken has published an article indicating that the mortality rate has been reduced to about 5 per cent. It might have been a relatively smaller series.

DR. ELLIS: Not in this one anyway. I would have thought that 10 per cent or 8 per cent was as much as he was claiming. This shows the

follow-up over a period of from six months to 36 months. The overall improvement of those who survived the operation was 55 per cent. Again, the rate of improvement decreased with length of follow-up. I think it is only fair to compare these results with what one would expect the outcome to be, had these patients been under medical treatment. In general, the average life expectancy of patients with symptomatic aortic stenosis is less than two years, and in the group we have followed it is shorter than that. It makes a great deal of difference what type of symptoms these people had at the time of surgery, because, certainly in our experience, the surgical mortality of patients that had been in unquestioned congestive heart failure is extremely high and the results have not, on the whole, been good, whereas the patients whose chief symptoms have been angina have been remarkably improved in a high percentage of cases. I might say, parenthetically, where pre- and postoperative catheterization studies have been done in various clinics, the degree of improvement is not well correlated with any measured change in the size of the valve judged by catheterizations done some months after surgery. For the most part, the improvement has been disappointing from the hemodynamic point of view.

MODERATOR KOSSMANN: Before we leave valvular lesions, we have a question from the audience. I wonder if any of the panelists can answer it. *What is the present status of multiple valve surgery? Dr. Johnson, have you any data?*

DR. JOHNSON: If you mean mitral and aortic valve surgery, we do have. We do just about as well with the patient with mitral stenosis and aortic stenosis as we do with one who has only mitral stenosis, when the patients are in the same state of symptomatology and cardiac size. That may be because the mitral valve is probably not as bad as it would otherwise be, since the two affected valves may combine to produce the symptom status. Since the beginning, we have been opening the mitral valve first, and then opening the aortic from below with the dilator, and have done quite well, we have thought, with that group. On the other hand, I don't actually have our figures on the longevity of these aortics, but I would guess that we have not done quite as well with them as with the mitrals, because my impression is that these patients with calcified aortic valves keep dropping off as time goes on, since the operation does not correct the original lesion of calcification of the valves. I don't really know, but I would guess that our results are

not quite as good as these at 36 months.

MODERATOR KOSSMANN: *What is the experience in Boston with multiple valve surgery in one sitting?*

DR. ELLIS: It is worth pointing out that the patient with both aortic and mitral disease resembles the mitral patient from the point of view of his clinical background. It is predominantly a disease of women just as is mitral stenosis. It occurs in the same group, and tends to run a long course. The calcification of the valve is frequently absent or minimal, as compared with the severe calcification of isolated aortic valve stenosis. Although I would not think that our results with the combined lesions are as good as in the pure mitrals, nevertheless they have done quite well.

DR. JOHNSON: I was thinking in terms of operative mortality rather than long-term results.

DR. ELLIS: The operative mortality is very low.

MODERATOR KOSSMANN: We have a few more minutes left, gentlemen, and I thought that we might just say a few quick words about surgery in coronary disease. The question I would like to ask is: *Is there any place at all in clinical medicine at the moment for direct or indirect surgery in coronary disease? By that I mean a direct attack on the coronary vessels themselves or the various indirect procedures that have been devised to increase the circulation to the myocardium. Dr. Ellis, do you have any views on this?*

DR. ELLIS: My personal experience has been limited to surgery for the relief of pain, that is, severe angina pectoris. I take a very dim view of the likelihood that we are improving the coronary circulation, or at least the nutrition to the heart, in any substantial degree by any surgical procedure. At least it has not been proven in human beings that this regularly happens. However, almost any of these operations will relieve the pain, at least for a considerable period of time, in a majority of patients. I don't know what the mechanism of relief is. It usually occurs promptly after the surgery, before one would think the collateral circulation would have time to develop. I think that in patients who have severe angina at rest, whose life is completely miserable and who are unable to do anything, any measure that relieves them of their pain is justified. In this group the use of these surgical measures, and I would take the mildest in preference to the more severe operations, is justified.

MODERATOR KOSSMANN: *Any preference among the milder opera-*

tions?

DR. ELLIS: By the mildest I mean putting powder into the pericardium, and perhaps tacking a bit of the lung over the heart. Actually, in two patients that we have recently examined post mortem, many months or years after such an operation, who had relief of their pain and who died of noncardiac causes, the chief circulation was through the lung, where the lingula was attached.

MODERATOR KOSSMANN: *Have the other panelists formed any opinions about coronary surgery?*

DR. HARVEY: No.

MODERATOR KOSSMANN: Dr. Johnson?

DR. JOHNSON: We have not been trying to "sell" anything in coronary surgery. We have really done very few of these operations, yet it is rather difficult to dismiss the subject completely. Dr. Beck has tried hard to popularize this procedure all over the country. He has succeeded better elsewhere than in his own home town. This has been rather disheartening for him. It is strange, when one operates upon these people, they are almost invariably grateful for having been operated upon and say that they are better. We just cannot evaluate it and say why they are better. The ones we usually operate on are those who say, "Well, if you don't operate on me, I am going over to New York and be operated on."

So I say, "Okay, I will do it, if that is the way you feel about it."

Actually, I have not done more than a couple of dozen of these altogether, but in that two dozen I don't believe there are more than two or three who have not thought that they were improved by the operation, and were very glad that it had been done.

MODERATOR KOSSMANN: I have some opinions on it, mostly indirect, having some familiarity with coronary disease. I have learned that this is an exceedingly capricious disease, and I have almost despaired of ever finding a way in which one could evaluate the usefulness or uselessness of any procedure designed for its treatment. I visualize the only way this can be achieved is to do a large number of cases such as reported by Dr. Ellis and Dr. Harken in rheumatic disease, and come up with a statistic. If this statistic is ever accumulated, we ought to run the series parallel, that is, have the control series run at the same time as the operated series and not try to compare an operated series done, let us say,

four or five years from now with a medical series observed five or ten years ago.

DR. JOHNSON: In all fairness, Dr. Beck has done just that. He has taken the patients that he operated upon, and the patients to whom he offered operation and who refused. His figures for the operated series are about twice as good as for those who refused operation, and the series includes a few hundred patients in each category. Still, it is hard to prove.

MODERATOR KOSSMANN: I would not disagree with that. I was in Cleveland recently, and after talking with the medical men there, my attitude and conclusions changed from what they had previously been as a result of examining the literature.

DR. JOHNSON: It is the medical men's figures that I am quoting. They are the ones who wrote the article. I would not, of course, accept his figures because he might be prejudiced!

MODERATOR KOSSMANN: That, of course, is a remote but real possibility. Ladies and gentlemen, our time is up. I would like to thank the members of the panel for their expert opinions and excellent data which they so generously presented to us this afternoon.